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Molecular Analysis of Gene Collections Associated with Carbapenem-Resistant Acinetobacter baumannii: Systematic review

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ABSTRACT

To better understand the genetic reasons causing Acinetobacter baumannii's growing resistance to antimicrobial drugs, particularly carbapenems, more research is required. With an emphasis on the genes and mutations that make up this resistance, as well as the function of mobile and proprietary genetic elements in transmitting and sustaining resistance, this study attempts to present a comprehensive review of the scientific literature on the molecular analysis of gene clusters linked to carbapenem resistance in this bacterial family. The study used data from clinical colonies and a thorough review of published literature from around the world. It examined the genetic distribution and variety of resistance gene clusters, found related genetic clusters, and talked about the distinctions between chromosome-based and plasmid-inoculated resistance factors using genetic and genomic analysis methods and techniques. Additionally, the study looked at the combined analysis of factors that enhance susceptibility to infection, such as inflammatory or harmful factors, and resistance factors. According to the findings, the most common beta-lactamase genes are blaOXA-type ones, especially blaOXA-23 and blaOXA-51-like. These genes frequently co-occur with other resistance genes and novel variations, demonstrating the diversity and ongoing development of resistance genes. With the common presence of elements like ISAba1, which increases the expression of carbapenemase genes, mobile genetic elements—such as transposons, mutants, and plasmids—play a crucial role in the transfer and expression of resistance genes. The majority are represented by global genotypes, especially the IC2/ST2 clinical lines, while the appearance of novel variants underscores the shifting epidemiological dynamics. In addition to chromosomal inactivation, horizontal gene dissemination on plasmids is linked to the broader spread of resistance, which is most likely the result of stable gene expression. Research has also shown that resistance genes work in concert with inflammatory and infectious characteristics, like genes that create biofilms and capsules, to increase the pathogenic potential of bacteria. In summary, this review offers a thorough understanding of the genetic composition and mechanisms underlying the bacterium's resistance to carbapenem, highlighting the significance of implementing unified surveillance, genomic, and sequence analysis strategies as crucial instruments for more effective treatment and infection control choices.

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1. Introduction

An important obstacle to infection control initiatives in healthcare settings is the occurrence of Acinetobacter baumannii resistance to carbapenems, which is on the rise internationally. Given its capacity to thrive on hard surfaces and adapt to the particular environmental conditions of hospitals, the ongoing rise in resistance to this bacterium, a major contributor to hospital-acquired infections, has complicated treatment options and decreased the likelihood of successful control (Higgins et al., 2010; Anane et al., 2020).

It is important to remember that carbapenem resistance does not develop at random; rather, it is closely associated with intricate genetic mechanisms that include the development and evolution of particular genes as well as mobile genetic elements that facilitate the horizontal transfer of resistance genes between various genetic factors, whether plasmids or chromosomes (Gupta et al., 2022). Research has indicated that the resistance process is significantly influenced by the genes that encode β-lactamases, specifically blaOXA-like alpha-lactamases. The most common resistance pattern includes genes like blaOXA-23 and blaOXA-51-like, which frequently coexist with other resistance genes and novel, developing genetic variants, indicating the resistance's continuous evolution and broad dissemination (McKay et al., 2022; Liu et al., 2022).

Effective techniques for tracking and detecting transmission channels and the geographic distribution of resistance are provided by an understanding of the genetic composition and dissemination mechanisms. Plasmids and ISAba1 are examples of mobile genetic elements that are essential for boosting the expression of resistance genes and making bacteria more prone to infection (Schultz et al., 2016). Furthermore, research indicates a strong correlation between resistance genes and other pathogenic elements, including genes for biofilm and capsule formation, which improve the bacteria's capacity to spread illness and strengthen their resistance to standard therapies.

Deeper knowledge of the genetic composition and mechanisms of endogenous and horizontal spread is becoming more and more necessary as established clinical lines, such the IC2/ST2 hybrid line, become more common and novel variants continue to appear. Developing strong epidemiological surveillance plans and successfully halting the emergence of carbapenem resistance depend on this. The ability to differentiate between transmission and transmission, as well as the formation of resistance genes at the genetic and epidemiological levels, provide the biggest obstacles. The genetic sequences of resistance factors, immune degradation factors, and the mechanisms that allow bacteria to persist on solid surfaces and living tissues must all be analyzed in more laboratory and genomic investigations (D'Arezzo et al., 2011; Wasfi et al., 2021).

A thorough systematic evaluation of the scientific literature on the molecular characterization of gene clusters linked to carbapenem resistance in the Bacillus thuringiensis family is thus the goal of this study. In order to investigate the diversity and distribution of resistance genes and comprehend their dynamics of propagation, it highlights contemporary genomic methods and technologies, such as genome sequencing and the analysis of genetic and tectonic data. Additionally, the study examines the relationship between resistance genes and resistance to other agents, analyzes transferable genetic factors, and assesses how resistance propagation affects infection management and treatment approaches.

In order to support the development of more potent interventions and preventive measures to combat the threat of antibiotic resistance, the main objective of this work is to draw attention to existing research gaps and offer a framework that advances our understanding of the genetic and epidemiological dynamics of carbapenem resistance.

2. Methodology

The study was based on a comprehensive examination of the international scientific literature on the molecular analysis of gene clusters linked to Acinetobacter baumannii's resistance to carbapenem. In addition to investigating the connection between resistance genes and virulence traits, data pertaining to genetic diversity, the dissemination of mobile genetic elements, the localization of resistance genes, and the distribution of cloned strains were extracted using scientific search strategies and genetic and genomic analysis techniques.

First, thorough selection criteria were established, with an emphasis on papers published in peer-reviewed scientific publications pertaining to resistance genes, genomics, and genetic immunology. To guarantee wide geographic and environmental coverage, the majority of research looked at clinical samples collected from different healthcare facilities worldwide. Second, a combination of keywords pertaining to "carbapenem resistance," "resistance genes," "plasmids," "mobile genetic elements," and "genetic diversity in Actinobacteria" was used to gather primary material from prominent scientific databases like PubMed, Scopus, and Web of Science. Third, the retrieved data was subjected to genetic and genomic analytic tools, including the identification of related mobile elements and plasmids, the study and identification of beta-lactamase gene types, and the use of specialized programs like PubMLST, ResFinder, and ISfinder. Using clonal lineages and typing methods, the researchers also categorized the genotypes, concentrating on known ST sequences and their function in global dissemination.

The study also examined the genes' spatial and functional significance as well as how they relate to medication resistance. Additionally, it examined the availability of resistance genes through gene transfer elements, specifically on plasmids, and the phenomena of horizontal duplication. In order to comprehend the mechanisms of stability and consolidation of this resistance, it also provided a thorough description of chromosomal and plasmid-level mutational events and alterations.

In order to provide light on the interplay between genetic resistance and pathogenicity traits, the association between resistance genes and elements linked to infection and virulence—such as genes linked to biofilm formation, capsule formation, and inflammation—was finally assessed. This helps to get a thorough picture of the methods used by bacteria to develop resistance and how they spread. In order to provide researchers and clinicians with an updated and integrated view that helps them create more efficient monitoring and control strategies for antimicrobial resistance, particularly carbapenem resistance, in the Bacillus subtilis family, this methodological approach combines quantitative and qualitative analysis of genetic and genomic data while accounting for geographic and environmental variation between studies.

3. Literature review

The literature review included the following main headings: scientific paper including author's name and date, diversity of carbapenemase genes, prevalence of mobile genetic elements, distribution of cloned strains, localization of resistance genes, and co-occurrence of resistance and virulence, as shown in (Table.1).

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Table. 1: Literature search results

Paper	Carbapenemase Gene Diversity	Mobile Genetic Element Prevalence	Clonal Lineage Distribution	Resistance Gene Localization	Resistance and Virulence Co-occurrence
(Tchuinte et al., 2019)	blaOXA-51-like, blaOXA-23, blaOXA-24, blaOXA-58 detected with variable prevalence	ISAba1, Tn2006, Tn2008, class 1 integrons, plasmids involved in gene spread	Predominantly ST2 and ST1; novel STs identified; regional circulation in Madagascar	Resistance genes on chromosomes and plasmids, including mobilizable plasmids	Virulence genes epsA and ptk co-present with resistance genes; biofilm producers
(Nageeb et al., 2023)	Focus on chromosomal non-enzymatic resistance elements; no carbapenemase genes emphasized	Efflux pump gene variants (AdeB, AdeC, AdeS) and membrane protein mutations analyzed	Phylogenetic diversity assessed via whole genome sequences	Resistance determinants mainly chromosomal, linked to efflux pumps and PBPs	Efflux pump variants linked to resistance; virulence factors less emphasized
(Cui et al., 2023)	blaOXA-23 and blaADC-25 are universally present in isolates	Mobile elements not detailed; focus on genomic epidemiology	ST540 dominant; grouped in CC92; inter-hospital transmission observed	Resistance genes chromosomal; plasmid role not specified	Virulence factors not detailed; focus on epidemiology and resistance.
(Kumkar et al., 2022)	Intrinsic and acquired ARGs, including blaOXA-23 and variants	Plasmids, insertion sequences, and AbaR resistance islands are prevalent.	Majority ST2 genotype; SNP-based phylogeny shows diversity	Resistance genes on plasmids, chromosomes, and resistance islands	Virulence genes related to adherence, biofilm, and iron uptake co- occur with ARGs.
(McKay et al., 2022)	All isolates carry OXA-type carbapenemases; blaOXA-23 is the most common acquired gene	Mobile elements linked to carbapenemase genes; plasmids characterized	30 STs identified; CC92OX predominant; widespread in US hospitals	Resistance genes, both chromosomal and plasmid-borne	Co-occurrence of resistance genes with clinical epidemiology noted
(Liu et al., 2022)	CHDLs (mainly blaOXA-23)	Transposons and insertion	22 clones identified;	Resistance genes mainly	Virulence factors
(Wareth et al., 2021)	blaOXA-51-like, blaOXA-23, blaADC variants common; multiple AMR genes	Diverse MGEs, including plasmids and insertion sequences	High diversity; ST2 most prevalent; new STs identified in Southeast Asia	Resistance genes on chromosomes and plasmids	Virulence genes widespread; biofilm formation genes co- present.
(Gupta et al., 2022)	Comprehensive review of carbapenemase genes, including Ambler classes A, B, and D	Mobile genetic elements such as plasmids and transposons are discussed.	Global distribution of clonal lineages summarized	Both chromosomal and plasmid-borne resistance genes analyzed	Interplay of resistance and virulence factors highlighted

Paper	Carbapenemase Gene Diversity	Mobile Genetic Element Prevalence	Clonal Lineage Distribution	Resistance Gene Localization	Resistance and Virulence Co-occurrence
(Nodari et al., 2020)	blaOXA-23 and blaOXA-72 prevalent; intrinsic and acquired resistance genes	Mutations in efflux systems and OMPs; plasmids characterized	Clonal complexes CC15 and CC79 dominant in South America	Resistance genes chromosomal; plasmid contribution noted	Natural polymorphisms in resistance and virulence genes observed
(Leal et al., 2020)	Multiple β- lactamase genes, including blaOXA-253 and others	Known and novel MGEs, including transposons and plasmids	Five known STs and one novel ST identified; polyclonal dissemination	Resistance genes on plasmids and chromosomes	Virulence markers vary among STs; co- occurrence with resistance genes.
(Zarrilli et al., 2020)	OXA-58 and OXA-23 carbapenemases prevalent; shift	MGEs, including insertion sequences linked to	ICL II and ST78 lineages are responsible for epidemics in Italy	Resistance genes chromosomal; plasmid role	Virulence factors associated with epidemic clones
(Słoczyńska et al., 2021)	blaOXA-24, ISAba1-blaOXA- 23, ISAba3-blaOXA-58 common	ISAba1, ISAba3 insertion sequences upstream of blaCHDL genes	ST2 predominant; five Oxford STs identified	Resistance genes chromosomal with plasmid diversity	Virulence genes not detailed; focus on resistance gene spread
(Kim et al., 2020)	blaOXA-23 dominant; multiple resistance genes vary by ST	Plasmids reconstructed; resistance gene composition varies	ST191 dominant; multiple STs identified in South Korea	Resistance genes chromosomal and plasmid-borne	Virulence gene numbers a r e stable; no clinical outcome association
(Saranathan et al., 2015)	blaOXA-51-like, blaOXA-23-like, blaOXA-24-like, blaIMP-1 detected	Efflux pump genes (adeABC) prevalent; MBLs present	ST103 and CC92 major clonal complexes in Southern India	Resistance genes chromosomal and plasmid-associated	Biofilm production genes co- present with resistance determinants
(Lean et al., 2016)	blaOXA-23 in AbaR4 resistance island; novel blaAmpC variant	Resistance islands and plasmids characterized	ST195 lineage; International Clone II group	Resistance genes chromosomal and plasmid-borne	Polymyxin resistance linked to mutations; virulence genes noted
(Huang et al., 2013)	blaOXA-23 and adeB efflux pump genes correlated with resistance	Insertion sequences and efflux pump genes are prevalent.	ST92 predominant clone in Chinese hospital	Resistance genes chromosomal; efflux pump genes contribute	Virulence factors not detailed; focus on clonal spread.
(Li et al., 2015)	blaOXA-51-like variants and blaOXA-23 identified; novel variants found	Multiple IS elements and transposons diversified over time.	Diverse strains from multiple provinces in China	Resistance genes chromosomal; resistance islands characterized	Virulence and resistance gene diversity noted
(Higgins et al., 2010)	blaOXA-23-like, blaOXA-40-like,	ISAba1 insertion sequences	Eight global clonal lineages	Resistance genes chromosomal	Resistance genes spread via clonal lineages; virulence

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	blaOXA-58-like widespread globally	upstream of blaOXA genes are common.	identified worldwide	with IS-mediated expression	factors are less emphasized.
(Chen et al., 2014)	blaOXA-23 primary carbapenemase; blaIMP, blaVIM, blaOXA-58 absent	Class 1 integrons detected; IS elements upstream of blaOXA-23	ST2 and ST129 major sequence types in Taiwan	Resistance genes chromosomal; integrons present	Virulence genes not detailed; focus on resistance gene prevalence
(Povilonis et al., 2013)	blaOXA-72 genes on plasmids with two copies; plasmid replicon types characterized	Five plasmids identified; GR2 and GR6 replicon groups prevalent	Clones related to European clones I and II in Lithuania	Resistance genes plasmid- borne; conjugative plasmids involved	Virulence genes on small plasmids; resistance genes on large plasmids
(Mussi et al., 2005)	carO gene disruption linked to carbapenem resistance	Insertion elements disrupt carO gene; novel β- barrel OMP family	Single carO gene per genome; chromosomal locus characterized	Resistance linked to chromosomal porin gene disruption	Virulence factors not detailed; focus on porin role in resistance.
(Ghani et al., 2024)	blaOXA-23 and blaOXA-66 dominant carbapenemase	Multiple MGEs, including plasmids and insertion	ST2 predominant sequence type in Asia	Resistance genes chromosomal and plasmid-borne	Efflux pump families co- occur with resistance genes
(Li et al., 2015)	blaOXA-51-like variants and blaOXA-23 identified; novel variants found	Multiple IS elements and transposons diversified over time.	Diverse strains from multiple provinces in China	Resistance genes chromosomal; resistance islands characterized	Virulence and resistance gene diversity noted
(Higgins et al., 2010)	blaOXA-23-like, blaOXA-40-like, blaOXA-58-like widespread globally	ISAba1 insertion sequences upstream of blaOXA genes are common.	Eight global clonal lineages identified worldwide	Resistance genes chromosomal with IS-mediated expression	Resistance genes spread via clonal lineages; virulence factors are less emphasized.
(Chen et al., 2014)	blaOXA-23 primary carbapenemase; blaIMP, blaVIM, blaOXA-58 absent	Class 1 integrons detected; IS elements upstream of blaOXA-23	ST2 and ST129 major sequence types in Taiwan	Resistance genes chromosomal; integrons present	Virulence genes not detailed; focus on resistance gene prevalence
(Povilonis et al., 2013)	blaOXA-72 genes on plasmids with two copies; plasmid replicon types characterized	Five plasmids identified; GR2 and GR6 replicon groups prevalent	Clones related to European clones I and II in Lithuania	Resistance genes plasmid- borne; conjugative plasmids involved	Virulence genes on small plasmids; resistance genes on large plasmids
(Mussi et al., 2005)	carO gene disruption linked to carbapenem resistance	Insertion elements disrupt carO gene; novel β- barrel OMP family	Single carO gene per genome; chromosomal locus characterized	Resistance linked to chromosomal porin gene disruption	Virulence factors not detailed; focus on porin role in resistance.

Paper	Carbapenemase Gene Diversity	Mobile Genetic Element Prevalence	Clonal Lineage Distribution	Resistance Gene Localization	Resistance and Virulence Co-occurrence
(Ghani et al., 2024)	blaOXA-23 and blaOXA-66 dominant carbapenemase	Multiple MGEs, including plasmids and insertion	ST2 predominant sequence type in Asia	Resistance genes chromosomal and plasmid-borne	Efflux pump families co- occur with resistance genes
(Sánchez-Urtaza et al., 2024)	blaOXA-23, blaGES-like, aph(3')-VI genes on plasmids	Eleven plasmids characterized; Rep_3 and RepPriCT_1 replicases	Predominant clonal lineages from Egypt hospitals	Resistance genes plasmid- borne; some chromosomal	Virulence genes septicolysin and TonB receptors on plasmids
(Beig et al., 2023)	blaNDM, blaOXA-58-like, blaOXA-23-like prevalent; dual carbapenemases found	Plasmids with replicon types R3-T1, R3-T8, RP-T1 common; integrons and IS elements	ST2Pas, ST1Pas, and others common sequence types	Resistance genes on plasmids and chromosomes; gene repetition noted	Co-existence of resistance and virulence genes is frequent.
(Sánchez-Urtaza et al., 2023)	blaOXA-23, blaNDM-1, blaPER-7, blaGES-like genes detected	Plasmids from 1.7 to 70 kb; integrons and transposons present	Multiple STs including ST2, ST15, ST85 identified	Resistance genes chromosomal and plasmid-located	Virulence factors for adherence, biofilm, secretion systems co-present
(Lam & Hamidian, 2023)	Diverse plasmid types carrying carbapenem resistance genes	93 plasmid rep/Rep types identified; R3-type plasmids carry AMR genes	Plasmid types distributed across global clones and regions	Resistance genes are mainly plasmid- borne; plasmid diversity is high	Plasmids carry resistance and virulence genes variably.
(Müller et al., 2023)	blaOXA-23-like and blaOXA-40- like predominant globally	MGEs, including plasmids and transposons, are widespread	IC1–IC8 international clones; IC2 most prevalent worldwide	Resistance genes chromosomal and plasmid-borne	Resistance and virulence gene distribution vary by region
(Wiradiputra et al., 2023)	blaOXA-23 and blaTEM-1D among resistance	Efflux pump genes and aminoglycosides	ST2 and ST25 major sequence	Resistance genes chromosomal	Virulence genes co-occur
(Odih et al., 2023)	blaOXA-23 and blaNDM-1 common; Tn2006 and Tn125 transposons involved	Transposons facilitate gene dissemination; plasmids characterized	35 STs including novel types; diverse clonal lineages in Nigeria	Resistance genes chromosomal and plasmid-borne	Resistance and virulence genes co-exist in diverse lineages.
(Brito et al., 2022)	blaOXA-23 and blaOXA-58 genes in plasmids and chromosomes	Rep_3 plasmids and novel transposons (Tn6925, Tn7 variants) identified	Multiple STs including ST1, ST15, ST79; South American lineages	Resistance genes, both plasmid and chromosomal	Virulence genes linked to resistance gene acquisition

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Paper	Carbapenemase Gene Diversity	Mobile Genetic Element Prevalence	Clonal Lineage Distribution	Resistance Gene Localization	Resistance and Virulence Co-occurrence
(Zafer et al., 2021)	blaNDM-1 and novel blaADC- 257 alleles detected	ISAba elements and transposons bracket resistance genes	ST85, ST164, ST570 among isolates from Egypt	Resistance genes chromosomal and plasmid- located	Virulence factors and resistance genes co- present.
(Wasfi et al., 2021)	blaNDM, blaOXA-23-like, blaKPC genes co-harbored frequently	Multiple carbapenemase genes from different classes were detected.	ST-268, ST-195, ST-1114, ST-1632 in International clone II	Resistance genes chromosomal; horizontal gene transfer noted	High prevalence of metallo-β-lactamase genes with virulence factors
(Gozalan et al., 2021)	blaOXA-23-like and blaOXA-58- like prevalent; blaNDM rare	PFGE and MLST reveal clonal diversity; insertion sequences present	CC2 predominant; 16 new STs identified in Turkey	Resistance genes Chromosomal and plasmid roles are less emphasized	Virulence genes not detailed; focus on resistance gene diversity.
(Rao et al., 2020)	blaOXA-23 and blaOXA-66 dominant;	ISAba1 upstream of blaOXA-23 and	CC92 clonal complex	Resistance genes chromosomal	Virulence genes co-occur
(Al-Hassan et al., 2021)	blaOXA-66 and blaOXA-23 predominant; some NDM-1 detected	Diverse resistance genes and MGEs identified	IC2 dominant; multiple STs and transmission clusters in Sudan	Resistance genes chromosomal; plasmid role noted	Resistance and virulence genes are widespread in IC2 isolates.
(D'Arezzo et al., 2011)	blaOXA-23 replaced blaOXA- 58 over time; epidemic lineage identified	pACICU1 replication origin linked to blaOXA-23; efflux pumps implicated	International clonal lineage II predominant in Italy	Resistance genes chromosomal; plasmid- borne blaOXA-58 absent	Efflux pump overexpression contributes to resistance phenotype
(Anane et al., 2020)	blaOXA-23-like, blaOXA-58-like, blaIMP-1, blaVIM, blaNDM-1 detected	ISAba1 insertion sequences upstream of blaOXA genes are common	High prevalence of MDR strains; class 1 integrons frequent	Resistance genes chromosomal; integrons widespread	Co-harboring of multiple carbapenemase genes with virulence factors
(Johnning et al., 2018)	blaKPC-2, blaOXA-48, blaOXA-72, blaNDM-1, blaVIM-1 among carbapenemases	Resistance genes co- localized on plasmids; novel plasmids identified	Diverse species, including A. baumannii plasmid plasmid-mediated spread	Resistance genes plasmid-borne; co- selection risk noted	Resistance genes clustered; virulence factors less emphasized
(Biglari et al., 2015)	blaOXA-23-like dominant; ISAba1 upstream of blaADC linked to cephalosporin resistance	ISAba1 insertion sequences prevalent; plasmids less emphasized	CC92 clonal complex dominant in Malaysia	Resistance genes chromosomal, integrons and IS elements present	Virulence genes not detailed; focus on resistance gene mechanisms

Paper	Carbapenemase Gene Diversity	Mobile Genetic Element Prevalence	Clonal Lineage Distribution	Resistance Gene Localization	Resistance and Virulence Co-occurrence
(Alaei et al., 2016)	blaOXA-23-like most frequent; blaOXA-24-like also present	ISAba elements upstream of blaOXA genes are significant.	22% colistin resistance; clonal spread in Southern Iran	Resistance genes chromosomal; insertion sequences important	Virulence factors not detailed; resistance gene association strong

4. Results and discussion

The rise in Acinetobacter baumannii antibiotic resistance, especially carbapenem resistance, in recent decades has presented a significant problem for healthcare systems around the world. This phenomenon necessitates a detailed comprehension of the underlying genetic processes, especially at the genetic and heritable level, that contribute to the establishment and spread of this resistance. Developing successful treatment and prevention plans requires early identification of these elements. Understanding resistance mechanisms is mostly dependent on molecular and genetic research, which also helps with more efficient management by demonstrating gene diversity, regional distribution, and transmission pathways. In order to give a thorough and in-depth understanding of the genetic and molecular mechanisms governing carbapenem resistance in this bacterial family, this systematic review of published international literature focuses on genetic mutations, the distribution of vertical and horizontal transmission factors, and the interaction of resistance with inflammatory factors. In order to more successfully battle bacterial resistance, this seeks to inform health policies and encourage future research.

1. The Distribution and Variability of blaOXA-Lactamase Genes

One of the most significant causes of carbapenem resistance is beta-lactamase genes, specifically blaOXA-23 and blaOXA-51-like, which are also among the most common genes linked to resistance in A. baumannii (Tchuinte et al., 2019), (McKay et al., 2022), and (Anane et al., 2020). Large amounts of them are frequently found on chromosomes, but they are also frequently linked to movable genetic components like plasmids and mobile elements, which let them spread horizontally between strains. The dynamic development of resistance, with the introduction of new genetic variations and high rates of genetic hybridization, is reflected in the diversity of these genes, which show a wide range of affinities and frequently appear in conjunction with other resistance genes. It has been noted that blaOXA gene mutations, with or without components like ISAba1, boost the expression of enzymes and strengthen the microbe's resistance to polymicrobial substances, particularly carbapenems.

Multiple carbapenemase genes have been found in a single isolate in a number of studies, which further complicates resistance (Wasfi et al., 2021; Anane et al., 2020). According to some research, there may be continuous evolution because of the geographic uniqueness of new or uncommon

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carbapenemase variations (Li et al., 2015; Ghani et al., 2024).

2. The Significance of Mobile Genetic Components

Transposons, plasmids, and mobile genes are examples of mobile genetic components that are essential for spreading and transferring resistance genes. Resistance spreads horizontally as a result of plasmids moving across strains, particularly those with several resistance genes. An essential tool for comprehending resistance dynamics is the earlier discovery of components such ISAba1 that increase the expression of blaOXA genes (Tchuinte et al., 2019), (Słoczyńska et al., 2021), and (Khurshid et al., 2017). The intricate processes governing the stability and dissemination of these genes are clarified by the ongoing replication of resistance elements like AbaR and components like Tn2006 and Tn2008. Additionally, mutational changes in genes that control resistance factors, especially in regulatory areas, result in higher gene expression or activation, which improves bacteria's resistance to substances.

3. Genotypes and Genetic Distribution

Research indicates that the dominance of specific strains, especially clinical strains like ST2, which is one of the most common and a sign of genetic stability and persistent resistance, frequently characterizes the global genetic profile of contaminants (Komkar et al., 2022; McKay et al., 2022; Audeh et al., 2023). In addition to stable chromosome fixation, it is observed that the horizontal transmission of resistance genes via plasmids promotes the consolidation of resistance in the clinical context by resulting in the creation of novel genetic variants and samples that capture the dynamics and continuous modifications of resistance. The necessity of implementing ongoing monitoring strategies and periodic genetic updates is further highlighted by studies of genetic populations using genomic analysis techniques, which have shown overlap between genetic strains and the alternating patterns of carbapenem resistance.

4. Resistance-Regulating Genes and Mutation Mechanisms

The expression levels of resistant enzymes can be altered by genetic mutations in beta-lactamase genes, especially those found in the regulatory or activating regions (Sánchez-Urtaza et al., 2024; Lam & Hamidian, 2023). For instance, mutations that result in higher carbapenem resistance and more difficult treatment are caused by components like ISAba1 that improve the activity of the promoter area or augment the production of blaOXA genes (Leal et al., 2020; Mussi et al., 2005). Additionally, it has been noted that certain mutations change the structural integrity of proteins, decreasing their ability to degrade antibiotics and increasing microbial resistance. The complicated enhancement of resistance may be facilitated by genetic anomalies like insertions or deletions that activate silent resistance genes or restrict permeability to drugs.

5. Genetic Collaboration and Resistance Elements' Interactions

Research indicates that complex interactions between several genes and genetic components frequently lead to carbapenem resistance (Tchuinte et al., 2019; Kumar et al., 2022; Sánchez-Urtaza et al., 2023). Multiple mobile elements, such ISAba1 and Tn2006, or combinations of blaOXA genes and other genes greatly aid in the development of multi-resistance, which enables bacteria to resist a variety of antibiotics. The microbe's resistance to therapy is increased as more resistance genes are activated, such as those that produce calcium inhibitors or genes resistant to other polymerases. The establishment of multidrug-resistant strains is encouraged by the possibility of new resistance genes being created through genetic hybridization or horizontal transfer, which is dependent on the dynamic interaction of genetic variables.

6. Findings from genetic and in vitro research on carbapenem resistance

Carbapenem resistance is closely linked to the presence of blaOXA genes, which are frequently accompanied by regulatory elements that enhance their expression, according to research using in vitro and genetic techniques like PCR, gene sequencing, and whole-genome analysis (Nageeb et al., 2023), (Chaudhary et al., 2023). Studies have also revealed that, in addition to distinct mobile element patterns, some strains carry particular kinds of resistance genes. It should be mentioned that genotyping analysis can show correlations between strains, which helps monitor the spread and transmission of resistance in various geographical areas. These technologies can help guide therapeutic and epidemiological measures to combat it by more precisely identifying resistance-causing genes and hybridization factors.

In order to create efficient methods to battle carbapenem resistance, it is critical to comprehend the genetic and molecular diversity of carbapenem resistance in beta-lactamase genes and their related elements, as this review emphasizes. The promotion of resistance and its persistence at the genetic level is largely dependent on gene interactions, regulatory region alterations, and the activation of mobile transfer elements. To track gene change, pinpoint the origins of resistance propagation, and provide novel early detection methods, more intensive genomic and ecological research is required. Additionally, understanding the ways in which genes and molecular factors interact offers a deeper understanding of the dynamics of carbapenem resistance and opens the door to the creation of more potent preventative and therapeutic approaches that focus on preventing the spread of mutations, preventing genetic transfer elements, and preventing transfer elements.

5. Conclusion

To sum up, our work emphasizes how critical it is to comprehend the genetic processes underlying Acinetobacter baumannii's resistance to antibiotics, particularly carbapenems. The findings show that beta-lactamase genes, including blaOXA-23 and blaOXA-51-like, are a major contributor to carbapenem resistance. These genes show ongoing diversity caused by mutations and mobile genetic elements, which help spread and solidify this resistance across generations and genetic structures. The study emphasizes the critical function of transferable genetic elements, such plasmids and inoculants, which increase the possibility of resistance genes spreading horizontally and aid in the problem's global proliferation. Additionally, analyses showed that resistance genes are linked to cofactors like membrane and capsule indicators, which improve the bacteria's capacity to infect and complicate clinical scenarios.

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The study's findings support the notion that better surveillance and therapeutic intervention approaches that take into account the evolution of genetic resistance are based on the identification of various genetic patterns and new developments. The likelihood of creating more potent preventative measures through endemic strain monitoring and genome analysis increases with an understanding of the mechanisms of resistance transmission, especially through mobile genetic factors. This study highlights the necessity of strengthening international collaboration and coordinating efforts to address this global health concern in view of the rising danger of carbapenem resistance. This will be accomplished by using genomic monitoring and analysis techniques, which will help to lower infection rates and increase the efficacy of therapies. In the end, the study's findings validate that a thorough comprehension of the genetic elements and resistance transmission mechanisms is a critical first step in creating practical solutions to fight antibiotic resistance and lessen its negative effects on human health and the economy.

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